

What Is Claimed Is:

1. A method of treating a hypoproliferative cell disorder or disorder involving increased cell death in a patient in need thereof, said method comprising administering to said patient a therapeutically effective amount of an EphA2 antagonistic agent.
2. The method of claim 1 wherein said hypoproliferative cell disorder or disorder involving increased cell death comprises the destruction, shedding, or inadequate proliferation of epithelial cells.
3. The method of claim 2 wherein said hypoproliferative cell disorder is interstitial cystitis or a lesion associated with inflammatory bowel disease.
4. The method of claim 1 wherein said hypoproliferative cell disorder or disorder involving increased cell death comprises the destruction, shedding, or inadequate proliferation of endothelial cells.
5. The method of claim 1 wherein said administration increases the proliferation or survival of an epithelial cell relative to the level of proliferation or survival in an untreated epithelial and/or endothelial cell.
6. The method of claim 1 wherein said administration increases the proliferation or survival of an endothelial cell relative to the level of proliferation or survival in an untreated epithelial and/or endothelial cell.
7. The method of claim 1 wherein said administration decreases EphA2 cytoplasmic tail phosphorylation relative to the untreated level of EphA2 cytoplasmic tail phosphorylation.
8. The method of claim 1 wherein said administration increases the integrity of an epithelial cell layer relative to the level of integrity of an untreated epithelial cell layer.
9. The method of claim 1 wherein said administration increases the integrity of an endothelial cell layer relative to the level of integrity of an untreated endothelial cell layer.

10. The method of claim 1 wherein said administration increases EphA2 gene expression or translation.
11. The method of claim 1 wherein said EphA2 antagonistic agent is an EphA2 polypeptide fragment comprising a ligand binding domain of EphA2.
12. The method of claim 1 wherein said EphA2 antagonistic agent is an antibody or antigen binding fragment thereof.
13. The method of claim 12 wherein said EphA2 antagonistic agent is an EphrinA1 antibody or antigen binding fragment thereof.
14. The method of claim 12 wherein the said antibody is a monoclonal antibody.
15. The method of claim 14 wherein said monoclonal antibody is a human antibody.
16. The method of claim 14 wherein said monoclonal antibody is humanized.
17. The method of claim 1 wherein said EphA2 antagonistic agent is chosen from the group consisting of a small molecule antagonist, enzymatic activity antagonist, EphrinA1 siRNA or eiRNA molecule, and EphrinA1 antisense molecule.
18. The method of claim 1 wherein said antagonistic agent increases EphA2 protein stability or protein accumulation.
19. The method of claim 1 wherein said administration decreases EphA2-endogenous ligand binding relative to the amount of untreated EphA2-endogenous ligand binding.
20. The method of claim 19 wherein said endogenous ligand is Ephrin A1.

21. The method of claim 1 further comprising the administration of one or more additional hypoproliferative cell disorder therapies that do not alter EphA2 expression or activity.

22. The method of claim 21 wherein said additional hypoproliferative cell disorder therapies consist of an immunomodulatory agent or an anti-urinary tract infection agent.

23. The method of claim 22 wherein said immunomodulatory agent is an antibody that immunospecifically binds IL-9.